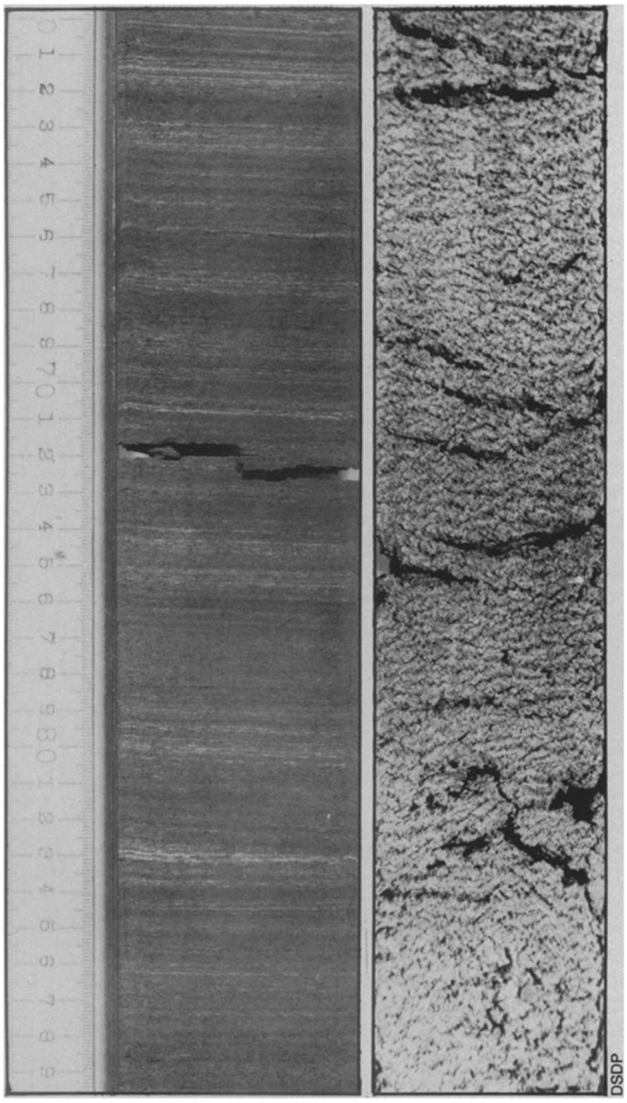
The best piston corer on the block



In striking contrast, these pictures pit the skill of a new piston corer developed at Scripps Institution of Oceanography (left) against that of a conventional rotary ocean drill (right). The new corer wins. First used on Leg 64 of the Deep Sea Drilling Project (SN: 2/10/79, p. 86), the Storms-Serocki Hydraulic Piston Corer allows scientists to recover the distinctly laminated sediments that are lost in the chewed-up jumble of rotary coring and yields cores more than 100 meters longer than other piston cores. Stan White of Scripps estimates, for example, that the upper 9 meters of a core, which are completely muddled by conventional coring, may represent as much as 10,000 years. The piston corer, by comparison, retains so much of the sediments' original character that light and dark varves (seasonal layers) are apparent. The lighter layers indicate periods when the waters were clear and when upwelling and winds carried nutrients, causing a diatom bloom. The darker layers mark the influx of rains, carrying clay and silt from land.

The two cores were taken at adjacent sites in the Gulf of California. These sections represent equivalent periods of time; the entire 152-meter piston core may extend as far back as 300,000 to 500,000 years. Researchers on Leg 68 plan to see just how deep the corer will go.

Cystic fibrosis: Genetic error found?

Cystic fibrosis, a disorder of the exocrine glands, is a common hereditary disease, especially among Caucasian populations. The malfunctioning glands produce a thick mucus that clogs the lungs and digestive system and can have fatal results in affected children. Now, the genetic defect that causes cystic fibrosis may have been found. Burton L. Shapiro and colleagues at the University of Minnesota in Minneapolis suggest that it is an abnormality in a cellular enzyme called NADH dehydrogenase.

The first clue came in 1977 when the researchers found that cystic fibrosis patients and carriers have excessive calcium between their cells. This excess was traced to the electron transport systems of the cells' mitochondria. Such an abnormality should result in excessive oxygen consumption in the cells of cystic fibrosis patients, and that's what the researchers found. They reported it in the June PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. When the researchers put an inhibitor of the electron transport system in the presence of cells from cystic fibrosis patients, the cells consumed the same amount of oxygen as did cells from healthy persons.

The inhibitor used by Shapiro and colleagues is known to have as its target NADH dehydrogenase, and the researchers suggest that the genetic defect in cystic fibrosis may involve that enzyme. In support of this hypothesis they report that NADH dehydrogenase differs in its activities depending on whether it is from cystic fibrosis patients, from carriers or from healthy subjects. The abnormal enzyme could result in excess calcium in exocrine gland cells, which in turn leads to abnormal mucus production.

These results must be confirmed, and an abnormal form or forms of NADH dehydrogenase must be identified in cystic fibrosis patients before the enzyme can be said to be involved in the disease. But if a faulty NADH dehydrogenase *is* indeed involved, it might eventually be possible to identify carriers. Both Shapiro and colleague Jim Peters, medical director of the Cystic Fibrosis Foundation in Washington, foresee this possibility. □

Insulin alchemy: Pig type to human

A bit of biological alchemy has converted pig insulin into human insulin. While some chemists are laboriously piecing together human insulin from its amino acid units and others are coaxing bacteria into production of insulin (SN: 9/16/78, p. 195), a group of Japanese biochemists have developed a simple insulin conversion technique. Insulin in both pigs and humans consists of 51 amino acids connected in two cross-linked chains. The pig and human varieties differ only in a single amino acid at the end of one chain. Scientists have attempted to chemically convert that amino acid, but the procedure was difficult because other parts of the molecule had to be protected against chemical change. Now Kazuyuki Morihara, T. Oka and H. Tsuzuki have used biological catalysts to more simply and economically make the conversion.

The scientists at Shionogi and Co., a large Japanese drug firm, used the enzyme carboxypeptidase to remove the final alanine from pork insulin. Then the enzyme trypsin was used to attach a threonine amino acid. The scientists were pleasantly surprised that the trypsin did not split other bonds in the insulin molecule. The final material was identified as human insulin by three chemical tests. Morihara and colleagues report in the Aug. 2 NATURE that the "semi-synthetic" human insulin is as active as pig or cow insulin in lowering blood glucose levels in experimental animals.

Human insulin is valued above the pig or cow hormone because scientists suspect that fewer human diabetics will have an allergic reaction to the human type. That suspicion, however, cannot be confirmed until sufficient human insulin is available for clinical trials. In the long run, converting pig insulin to the human variety does not overcome the problem of a limited insulin supply for the steadily increasing population of diabetics. Such conversions might just exacerbate the shortage because the conversion reported only gives a 41 percent yield. For the near future the recombinant DNA technique is the one most likely to provide an insulin supply independent of the climatic conditions, animal prices and vagaries of the meat industry that now affect insulin availability. □